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AGING OF THE MYOCARDIUM*

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THERE are so many examples of a contrast between apparent age and the conservation or loss of function that we should not be surprised that this contrast is great in the myocardium. A bald or white-haired man, with wrinkled face and store-bought teeth, may turn out to be a superlative chess-player or contributor to scientific progress, and the wizened, dark-brown heart of a 90 year old man may have beaten regularly and maintained an adequate circulation as he died of cancer. On the other hand, a good-looking young man may turn out to be the superannuated baseball hero of the previous decade, no longer able to hit or run, and the fine, large, pink heart may be that of a woman 55 years old, who died of congestive heart failure and auricular fibrillation, with no valvular disease or hypertension.

With aging, all hearts accumulate pigment granules, which are concentrated close to the nuclei. If the heart muscle remains efficient, and therefore can atrophy as the demands of basal metabolic load, digestion and physical activity decline with advancing years, the ratio of pigment to myoglobin and myofibrils is high and we see a small

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brown heart. On the other hand, if the myocardium becomes inefficient, if more muscle mass is needed for each unit of work, or if the load imposed by valvular disease, hypertension, or high volume flow of blood leads to hypertrophy, then the ratio of pigment to myoglobin and myofibrils is low. Microscopically as well as grossly, the inefficient or overloaded heart looks more normal in the aged, than the efficient muscle carrying its small load.

In addition to changes in the pigment content, and the diffuse atrophy of senile inanition, the hearts of obese sedentary Americans often show, even before 50, striking local atrophy and even complete disappearance of myocardial fibers in the right ventricle and right atrium. In these hearts, yellow adipose tissue, continuous with the abundant epicardial fat, or developing *de novo* under the endocardium, completely replaces myocardial tissue. In extreme examples, much of the right atrium near the annulus looks like pure fat when sectioned.

There is no doubt whatever that the aging heart can beat efficiently in most people as long as the coronary system is not damaged by a disease which in itself is not a part of the aging process. Even chronic valvular disease and hypertension, or the high flow imposed by parkinsonism or by Paget's disease, may fail to produce a dilated and failing heart in a man of 70 or more. But statistics, rather than the outstanding exception, must be used in approaching such a problem, and the figures show clearly that with any type of injury or embarrassment to the heart, dilatation and failure occur far more frequently after 50 than before 30 years of age. Army personnel dying of coronary disease before 40 had little heart failure, and no right ventricular failure at all. Those dying of coronary disease after 55 years of age exhibited left heart failure in over 50 per cent, right heart failure in nearly 20 per cent of cases. Heart failure and auricular fibrillation are frequent even in mild hyperthyroidism after 50; they are unknown, even in severe Graves' disease, before 30. Severe hypertension rarely causes heart failure in young people; mild hypertension often is the only demonstrable circulatory embarrassment in elderly cardiac invalids. Nor is it merely the tendency to auricular fibrillation and congestive failure which distinguishes old hearts from young ones. Young people with complete heart block rarely have the syncopal attacks, due to ventricular stand-still, which so often occur in elderly people with heart block. The old heart has neither as dependable a set of stand-by pacemakers,

as dependable machinery for conducting the normal impulse and suppressing ectopic foci, nor as efficient a mechanism for converting chemical to contractile energy, as did the same heart a few decades earlier in life.

It is not known what differences actually exist between young and old hearts in the various factors which could alter the irritability and efficiency of the muscle, but perhaps it is worth while to state what some of these factors could be. The work of Szent-Györgyi and of Salter showed that normal plasma contains a steroid which increases myocardial efficiency, but the level of this substance in the plasma of young and old, and the responsiveness of young and old hearts to given levels of the steroid are unknown. The ionic balance of sodium, magnesium, calcium, and potassium in the fluid about the myofibrils is known to alter myocardial efficiency; the effect of age on this balance is unknown. The proteins of the myofibrils, actin and myosin, and the adenosine phosphates of muscle react with each other to cause contraction and relaxation, but the effects of aging on these reactions are unknown. Finally, the mechanism by which lactate, pyruvate, dextrose or fat are burned and provide energy to the contractile system may be altered by age, but no data on this have been obtained. Any or all of these may be affected by the aging process, and the change might involve either the amount of energy available for a contraction after a long rest interval, or merely the rate at which capacity for maximal contractile efficiency was restored after each beat.

The clinical impression is that the greatest change caused by aging of the myocardium is delay in recovery of contractility and irritability. It is known that most arrhythmias are due to delay in recovery of irritability, with prolongation of both relative refractory and supranormal phases of irritability facilitating the origin of ectopic beats or rhythms. The older hearts are much more prone to these disorders. In young people, ventricular rates over 200 per minute may last for many hours or even days before the heart dilates and signs of heart failure set in. In people over 60, heart failure is rare with complete heart block or in the fevers with bradycardia, such as typhoid. But rates of 120 to 150, due to paroxysmal tachycardia or to fever, frequently precipitate failure in older people. It would therefore appear that the old myocardium has a slow rate of recovery, but contracts well as long as there is an adequate period for rest between beats. This is why simple

emotional tachycardia, with no rise in blood pressure, so often seems to be a factor in precipitating acute heart failure in people over 50.

Ever since heart failure was precipitated by overdosage of desoxycorticosterone, in treating Addison's disease, it has been realized that excessive sodium ingestion or retention led to heart failure not only by hastening edema formation, but by actually impairing myocardial efficiency. Much experimental and clinical evidence has confirmed an idea that goes back to the observations of Ringer on the effect of electrolytes on the heart beat. There is an ideal balance of metallic ions, at which the myocardium functions best, and its efficiency is impaired when this balance is altered. Deficits of potassium and excesses of sodium seem to be particularly bad for the heart. The former is frequent with severe illness, and in the diabetic; the latter is the normal state of civilized man, with a daily sodium to caloric intake from 10 to 30 times that of his anthropoid cousins and 5 to 10 times that of primitive races or the sweating masses of poor people throughout most of the world today. Thus, it is difficult to be sure that the loss of myocardial efficiency with age is actually due to an involutional process, and not the cumulative result of decades of injury due to excessive salt intake and a sedentary existence, with low salt need. Perhaps the best evidence for the view that aging alone can impair myocardial function is that auricular fibrillation and heart failure occur in old horses, which have not been on high salt diets and have faultless coronary arteries.

It should be emphasized that none of these anatomical or chemical changes impair the ability of the myocardium to respond to modest loads of work; none of them actually cause heart failure. All of them reduce the ability of the heart to meet sudden increases in rate or in work per beat, and thus they predispose the heart to fail during febrile illnesses, after operations or pulmonary embolism, or with the chronic causes of cardiac strain—Parkinson's disease, Paget's disease, leukemia, anemia, and hyperthyroidism—which so often occur in elderly people and raise the pulse rate and the stroke volume of the heart. Pulmonic or systemic hypertension, also common in aging Americans, brings to light the inability of old hearts to cope with a heavy burden.

Whatever the background of failure of the myocardium, this type of disorder responds very well to digitalis and to salt restriction. Unfortunately, elderly people are often "set in their ways" and do not take kindly to diets of rice, green vegetables, salt-free cheese and

boiled meat. As Kempner and others have shown, the cardiacs who do adhere to such diets, with sodium contents comparable with those in the natural diets of the great apes, show striking benefit even without digitalis. But most of our patients prefer a less physiologic therapeutic program, based on mercurial diuretics, digitalis and Diamox. It is unfortunately true that just as no synthetic institutional program can ever substitute for a large and affectionate family in providing the proper environment for an aging man or woman, no program of drugs ever will provide a satisfactory replacement for a physiologic regime of activity and diet, in an atmosphere of pure air. The elderly cardiac probably fared better at Cos or Epidauros, in the hands of Hippocrates and his disciples, than he does in the smoke-filled waiting rooms of our doctors' offices and with the powerful therapeutic agents available today.

The most encouraging thing about myocardial aging is that the rate of progress often is slow in those who develop symptoms because of intercurrent illness such as myocardial infarction, if a sound program of digitalization and sodium restriction or depletion is adhered to. We have seen patients with excellent capacity for physical effort ten, and in two cases, twenty years after their initial bouts of acute pulmonary edema. This can only happen when the physician and the patient refuse to accept the idea that aging of the heart (presbycardia) or what has been called arteriosclerotic heart disease, is a relentlessly progressive disorder which of itself is fatal.

As with what is called cerebral arteriosclerosis, the aging changes in the heart can be imitated by vitamin deficiency and endocrine imbalance. Until all these possible causes or potentiators of dysfunction have been treated or completely excluded, it is improper to put the blame on involutional change. Just as a mind which might continue to function adequately in a calm home, with old friends and affectionate relatives, will seem to disintegrate in a hospital, a nursing home, or a psychiatric ward, so a heart which would give no trouble on a good diet and in a tranquil person rapidly fails during periods of social or family stress, with diets high in sodium and low in water-soluble vitamins. Aging of the heart or mind do least damage and progress very slowly when the individual lives under optimal conditions, and the manifestations seen under other situations can be corrected and further symptoms prevented when the patient receives and accepts sound and optimistic advice.